ON-LINE CASE REPORT

Ann R Coll Surg Engl 2010; **92**:
doi 10.1308/147870810X1269966298091

Traumatic arterial spasm causing transient limb ischaemia: a genuine clinical entity

G Peach[1], GA Antoniou[1], K El Sakka[1], M Hamady[2]

[1]Regional Vascular Unit and [2]Department of Interventional Radiology, St Mary's Hospital, Imperial College Healthcare Trust, London, UK

ABSTRACT

Traumatic arterial spasm is a phenomenon that has long been questioned by clinicians. Indeed, some would argue that surgical exploration is mandatory whenever there are signs of distal ischaemia following limb trauma. We present a case of angiographically demonstrated tibial artery spasm following gunshot injury. Exploration was unnecessary and distal perfusion was re-established spontaneously. This case demonstrates the existence of traumatic arterial spasm as a genuine clinical entity and suggests that immediate surgical exploration may not be necessary in all cases.

KEYWORDS

Arterial spasm – Trauma – Ischaemia – Gunshot

Accepted 18 May 2010; published online 23 June 2010

CORRESPONDENCE TO

George Peach, Regional Vascular Unit, St Mary's Hospital, Imperial College Healthcare Trust, London, UK
E: george_peach@hotmail.com

Many authors question the existence of traumatic arterial spasm and advocate urgent surgical exploration whenever traumatic limb injury is associated with signs of ischaemia.[1] We present a case of trauma-related limb ischaemia that was successfully managed conservatively following angiographic identification of arterial spasm rather than true arterial injury.

Case history

A 31-year old man was admitted to the emergency department after sustaining two gunshot wounds to his left leg. The shots had been fired from a small calibre hand-gun, the first bullet passing through the medial part of his left thigh without causing injury to bone or any large vessel. The second bullet had entered the medial side of his distal leg, resulting in a long, oblique fracture to the upper third of the tibia, with associated fracture of the fibular neck without obvious external limb deformity. No other injuries were identified and the patient was haemodynamically stable.

On examination of the injured limb, there were signs of acute ischaemia. No pedal pulses could be felt and the foot was pale and cool with slowed capillary refill. Furthermore, there was no detectable Doppler signal at the ankle. There was no evidence of significant contained haemorrhage within the limb and only minor active bleeding from entry and exit wounds. Movement and sensation were intact.

When angiography was performed shortly afterwards to identify the site of the expected arterial injury, all vessels

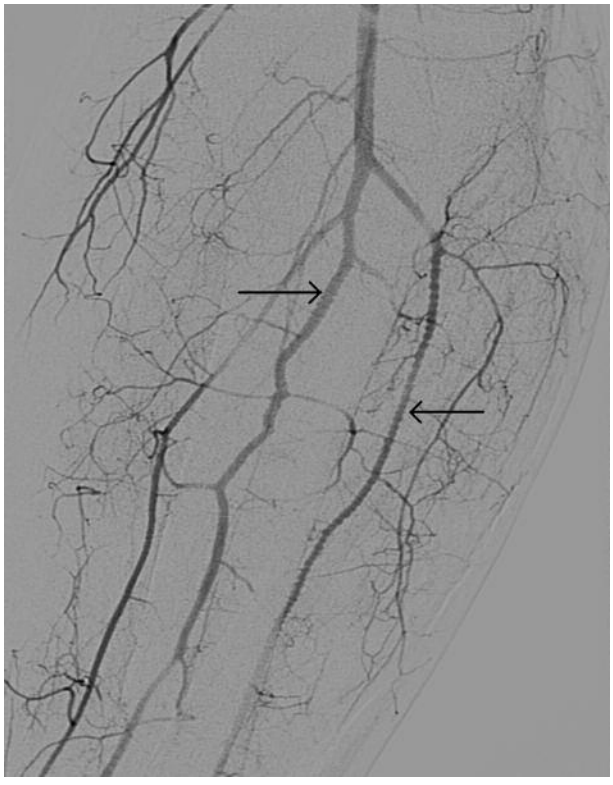

Figure 1 Digital subtraction angiography at the level of the proximal left leg shows intact arterial tree. Note the diffuse beaded appearance of the anterior tibial and peroneal arteries (black arrows). The posterior tibial and popliteal arteries are normal.

were found to be intact. There was, however, flow-limiting vasospasm of all three tibial vessels, particularly the anterior tibial and peroneal arteries (Fig. 1). In light of these findings (and as the leg was not critically ischaemic), it was decided to manage the spasm conservatively, with regular observation and a plan for urgent intervention should the ischaemia progress.

Within 2 h, he was taken to theatre for fixation of the tibia and foot fasciotomies. When distal limb perfusion was re-assessed just prior to orthopaedic intervention, there was no longer any evidence of ischaemia and full foot pulses were readily palpable in the affected limb with triphasic Doppler signals. At follow-up 2 months later, the injured limb was well perfused.

Discussion

Vasospasm of small or medium size arteries such as coronary, carotid and cerebral arteries has been well documented in the literature, with many possible stimuli proposed. These stimuli include toxins (*e.g.* ergot alkaloids or cocaine), endothelial damage (*e.g.* from external trauma or venepuncture), cold or emotion (*e.g.* Raynaud's phenomenon), and excessive release of intrinsic vasoconstrictors (*e.g.* histamine or prostaglandins).[2] However, there remains much contention over the existence of trauma-induced arterial spasm in the absence of arterial wall damage. Some authors have proposed that it may arise as a result of external mechanical stimulation caused by bone fragments or transmitted shockwaves.[3]

Uncertainty about the condition has led to the widely held view that all patients who have sustained injury to an extremity should undergo urgent surgical exploration if there are any objective signs of limb ischaemia. Furthermore, it has been suggested that distal ischaemia following limb injury should never be attributed to transient vasospasm in the hope that it might resolve spontaneously: this simply delays operative revascularisation and results in greater long-term morbidity.[4]

In contrast to such a generic management plan, a case of spontaneously resolving traumatic arterial spasm is described here. Despite clear signs of ischaemia and initial absence of foot pulses, good arterial flow returned spontaneously within a relatively short time and certainly well before there was risk of long-term anoxic tissue damage. It is important to note that there was significant restoration of perfusion before fracture reduction was performed, suggesting that flow recovery was not simply due to removal of extrinsic compression or correction of arterial displacement. We, therefore, propose that objective diagnostic measures such as digital subtraction angiography or computed tomography (CT) angiography should be used whenever limb trauma is associated with ischaemia – assuming there is no overt arterial injury requiring immediate surgical management. This might allow unnecessary surgical intervention to be avoided.

In our case, arterial spasm (most probably due to widespread kinetic energy dispersion caused by the gunshot impact) produced a concertina-like appearance on angiography, with multiple contractions of the arterial wall. This has rarely been demonstrated and other authors have described angiographic images that mimic true vascular injury, with tapering of the artery and no intimal irregularity. Subsequent expert radiological review of our images excluded the possibility of artefact due to 'contrast beading'.

In this case, it was possible to manage the patient conservatively as there were no signs of limb-threatening ischaemia. However, if vasospasm of this type can be correctly identified, it may be possible to use non-operative measures to restore distal perfusion even if signs of limb-threatening ischaemia do develop. A number of endoluminal options have been proposed for treatment of spasm in small- or medium-sized arteries, most commonly involving use of intra-arterial vasodilators such as calcium channel antagonists, nitrates and papaverine. It has also been shown that in instances of more localised arterial spasm, balloon angioplasty of the affected vessel can be effective in re-establishing arterial flow.[5]

Conclusions

This case highlights the importance of considering arterial spasm as a cause of limb ischaemia after trauma. With timely use of appropriate imaging, it may be possible to identify the condition accurately and avoid unnecessary operative intervention.

References

1. McCoy G, Hannon D, Barr R, Templeton J. Vascular injury associated with low-velocity dislocations of the knee. *J Bone Joint Surg Br* 1987; **69**: 285–7.
2. Majno G, Joris I. *Cells, tissues, and disease: principles of general pathology.* Oxford: Oxford University Press, 2004.
3. Haug, R. Management of low-caliber, low-velocity gunshot wounds of the maxillofacial region. *J Oral Maxillofac Surg* 1989; **47**: 1192–6.
4. Lord RS, Irani CN. Assessment of arterial injury in limb trauma. *J Trauma* 1974; **14**: 1042–53.
5. Hoh B, Ogilvy C. Endovascular treatment of cerebral vasospasm: transluminal balloon angioplasty, intra-arterial papaverine, and intra-arterial nicardipine. *Neurosurg Clin North Am* 2005; **16**: 501–16.